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Effects of noxious stimulation to the back or calf muscles on gait stability

Wolbert van den Hoorn^{a,*}, François Hug^{a,b}, Paul W. Hodges^a, Sjoerd M. Bruijn^{c,d},
Jaap H. van Dieën^c

^a The University of Queensland, Centre for Clinical Research Excellence in Spinal Pain, Injury and Health, School of Health & Rehabilitation Sciences, Brisbane, Queensland 4072, Australia

^b University of Nantes, Laboratory "Motricité, Interactions, Performance" (EA 4334), Nantes, France

^c MOVE Research Institute Amsterdam, Department of Human Movement Sciences, VU University Amsterdam, Amsterdam, The Netherlands

^d Department of Orthopaedic Surgery, First Affiliated Hospital, Fujian Medical University, Fuzhou, Fujian, PR China

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ABSTRACT

Gait stability is the ability to deal with small perturbations that naturally occur during walking. Changes in motor control caused by pain could affect this ability. This study investigated whether nociceptive stimulation (hypertonic saline injection) in a low back (LBP) or calf (CalfP) muscle affects gait stability. Sixteen participants walked on a treadmill at 0.94 ms⁻¹ and 1.67 ms⁻¹, while thorax kinematics were recorded using 3D-motion capture. From 110 strides, stability (local divergence exponent, LDE), stride-to-stride variability and root mean squares (RMS) of thorax linear velocities were calculated along the three movement axes. At 0.94 ms⁻¹, independent of movement axes, gait stability was lower (higher LDE) and stride-to-stride variability was higher, during LBP and CalfP than no pain. This was more pronounced during CalfP, likely explained by the biomechanical function of calf muscles in gait, as supported by greater mediolateral RMS and stance time asymmetry than in LBP and no pain. At 1.67 ms⁻¹, independent of movement axes, gait stability was greater and stride-to-stride variability was smaller with LBP than no pain and CalfP, whereas CalfP was not different from no pain. Opposite effects of LBP on gait stability between speeds suggests a more protective strategy at the faster speed. Although mediolateral RMS was greater and participants had more asymmetric stance times with CalfP than LBP and no pain, limited effect of CalfP at the faster speed could relate to greater kinematic constraints and smaller effects of calf muscle activity on propulsion at this speed. In conclusion, pain effects on gait stability depend on pain location and walking speed.

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1. Introduction

Stable walking without undue fall risk requires appropriate control to deal with perturbations that originate in the external environment and inside the body (Bruijn et al., 2013; Toebes et al., 2012). As a result of impaired sensorimotor control, reduced gait stability is associated with aging (Kang and Dingwell, 2008) and neurological disorders (Ijmker and Lamothe, 2012; Reynard et al., 2014). Musculoskeletal pain has been associated with fall risk (Asai et al., 2015; de Zwart et al., 2015; Kitayuguchi et al., 2015) and may also impact stability. For example, spinal movement stability was lower during pain than no pain (Ross et al., 2015). It is important to address the effects of pain on gait stability as musculoskeletal pain increases with age (Hoy et al.,

2014; Smith et al., 2014), which potentially could increase falls risk (Foley et al., 2006; Leveille et al., 2009).

Motor adaptations to pain are thought to protect the painful/injured tissues (Hodges and Tucker, 2011; Lund et al., 2011; van Dieën et al., 2003). Such adaptations are thought to increase joint stiffness (Hodges et al., 2013; van den Hoorn et al., 2012) and could potentially enhance stability. However, increased stiffness may coincide with reduced responsiveness. In addition, nociceptive input may impair proprioceptive acuity (Brumagne et al., 2000; Lee et al., 2010; Matre et al., 2002) and force regulation (Descarreaux et al., 2005; Salomoni et al., 2013) which could reduce stability. The effect of pain on gait stability is likely to depend on the region that is painful and, if muscle is painful, on its biomechanical role in gait. The calf muscle–tendon unit is thought to be the principal contributor to propulsion/push-off, but also to contribute to frontal plane movements (Kim and Collins, 2015; Pandey and Andriacchi, 2010). Back muscles control trunk orientation by counteracting movements, such as those induced by push-off and

* Corresponding author. Tel.: +61 7 3365 1355; fax: +61 7 3365 1284.

E-mail address: w.vandenhoorn@uq.edu.au (W. van den Hoorn).

heel-strike. Because of these distinctive biomechanical roles of calf and back muscles, nociceptive irritation of these muscles may affect gait stability differently.

The study aim was to determine the effects of experimental nociceptive stimulation of the calf (medial gastrocnemius) or back (lumbar erector spinae) muscles on gait stability. Pain was induced by intramuscular injection of hypertonic saline, which activates muscle nociceptors without changing muscle properties. Gait stability was assessed with the local divergence exponent (LDE) of thorax movements (Bruijn et al., 2009a; Dingwell et al., 1998; Rosenstein et al., 1993), which assesses the system's sensitivity to small perturbations. Because gait stability is affected by speed (Bruijn et al., 2009a; Dingwell and Marin, 2006), we compared two walking speeds; 0.94 ms^{-1} and 1.67 ms^{-1} . The magnitude and stride-to-stride variability of thorax movements, which relate to the magnitude of gait perturbations, and the temporal gait parameters were also assessed. We hypothesized that: (1) motor adaptation induced by pain would lower gait stability and increase variability; and (2) noxious stimulation of the calf would have greater impact on gait stability than of the back muscles because of the more critical role of calf muscles for gait.

2. Methods

2.1. Participants

Seventeen healthy volunteers (6 females and 11 males; mean \pm standard deviation, age: 21 ± 2 years, weight: 66 ± 11 kg, height: 173 ± 10 cm) were recruited via advertisements on the university student website. Participants had no history of back or lower limb pain that limited normal function or required them to seek intervention from a health care professional. Participants provided written informed consent. The Institutional Medical Research Ethics Committee approved the study, and procedures conformed to the Declaration of Helsinki. One participant fainted during preparation, thus, data are reported for 16 participants.

2.2. Experimental setup

To minimize perturbations induced by the external environment, experiments were conducted on a treadmill (BH fitness, Pioneer pro, Spain) at 0.94 ms^{-1} and 1.67 ms^{-1} . Reflective markers (14 mm) were attached to the skin with double-sided tape according to the Vicon Plug-in-Gait marker set. Marker positions were recorded using an 8-camera system at 100 sample/s (T040, Vicon Motion Systems Ltd, Oxford, UK). The global X-axis was aligned with the participants' walking direction, Y-axis to the left and Z-axis upwards. For a separate study, myoelectric activity of 19 lower leg, upper leg and trunk muscles on the right side was recorded (see van den Hoorn et al. (2015)).

2.2.1. Procedure

Participants walked barefoot and familiarized with treadmill walking for 1–5 min before the start of the experiment. Walking trials were repeated in 5 experimental conditions: control (control); low back pain (LBP); washout LBP; calf pain (CalFP); and washout CalFP. Each trial included 3 min of walking at 0.94 ms^{-1} and at 1.67 ms^{-1} . Order of both speed and pain location was balanced. All participants began with the control condition, which was considered the reference for both pain conditions. Participants rested in sitting between conditions and speeds for ~ 5 min. Washout conditions began ~ 4 min after complete resolution of pain.

2.2.2. Experimental pain

To induce acute muscle pain, a single bolus (0.7 mL, 7% NaCl) of hypertonic saline was injected into the right erector spinae muscle adjacent to the L3 spinous process or the middle of the muscle belly of the right medial gastrocnemius muscle. Each participant received 4 hypertonic saline injections; one at each walking speed for each location. The pain intensity was reported verbally every 30 s during the painful conditions on an 11-point numerical rating scale, anchored with “no pain” at 0 and “worst pain imaginable” at 10.

2.3. Data analysis

Data analysis was performed in Matlab (The Mathworks, Inc., Natick, MA, USA). For the pain trials, the start of the time section was selected after pain intensity was reported above 3/10. The number of strides included in the analysis was capped at 110 to ensure that all participants were represented by the same number of strides

and still had a pain intensity above 2/10 at the end of this time section. For the control and washout conditions the first 110 strides were analyzed.

2.3.1. Temporal gait parameters

Heel strikes were determined from the local vertical minima of the heel marker position, and toe-off was determined from the local vertical maxima of the heel marker velocity (Pijnappels et al., 2001). *Stride time* was the time between consecutive heel strikes of the right leg. *Stance time* was the time between heel strike and the consecutive toe-off on the same side. *Swing time* was the time between toe-off and heel strike on the same side.

2.3.2. Pre-processing of kinematic data

Thorax movements along the anteroposterior (AP, X-axis), mediolateral (ML, Y-axis) and vertical (VT, Z-axis) axes in relation to the external Vicon reference frame were used to calculate gait stability. Data were left unfiltered (Mees and Judd, 1993). Thorax was represented by the average positions of the clavicle, sternum, and T10 markers. To avoid non-stationary data, position data were differentiated over time to obtain velocity.

2.3.3. Gait stability (local divergence exponents; LDE)

LDE estimates are biased to number of samples present in time series (Bruijn et al., 2009b). For example, with fewer samples in the time series due to shorter stride times, nearest neighbors tend to be further apart and consequently initial divergence tends to be slower. Therefore, each time series containing 110 strides was time normalized using spline interpolation to a fixed number of samples (11,690) while retaining between stride variability.

For each movement direction, a five dimensional state space was reconstructed with delay embedding of the thorax velocity signals (Takens, 1981). The number of dimensions was derived from false nearest neighbor analysis (Kennel et al., 1992). The delay for embedding was fixed to 10 samples for each participant, as each stride cycle contained a similar number of samples (Bruijn et al., 2009b; England and Granata, 2007; van Schooten et al., 2012). Divergence curves were calculated according to Rosenstein (Bruijn et al., 2009b, 2009a; Rosenstein et al., 1993). The divergence of nearby trajectories in state-space was expressed by the Euclidian distance between trajectories starting at nearest neighbors. To obtain the mean logarithmic divergence rate, the average was calculated across all log-transformed original nearest neighbor trajectories. The LDE was then determined as the slope of the linear regression of the average logarithmic divergence between 0 and $\frac{1}{2}$ stride cycle (Bruijn et al., 2009b, 2009a; Stenum et al., 2014).

2.3.4. Magnitude and variability of thorax movement

The magnitude and variability of thorax velocities along the AP, ML and VT axes, were assessed by the RMS values and the stride-to-stride variability, respectively. For analysis of the stride-to-stride variability, data were time normalized to stride cycle duration (from right heel strike to following right heel strike) with a 101-point spline interpolation. At each stride cycle percentage, the standard deviation over all strides was calculated, then the median of the 101 standard deviations quantified the stride-to-stride variability magnitude (SDs).

2.4. Statistical analysis

Statistical analyses were performed in Stata (v12, StataCorp, College Station, TX, USA). Significance level was set at $P \leq 0.05$. Repeated measures analysis of variance (ANOVA) was used to test the effect of pain on the outcome measures. For analysis of LDE, RMS and SDs, Condition (control, LBP, washout LBP, CalFP, washout CalFP), speed (0.94 ms^{-1} and 1.67 ms^{-1}) and movement axis (AP, ML, and VT) were entered as within subject factors. For analysis of stride time, condition and speed were entered as within subject factors. For analysis of stance time and swing time, condition, speed and side (left and right) were entered as within subject factors. For analysis of pain level, pain Location (calf and back) and speed were entered as within subject factors. Post-hoc analyses were applied with Bonferroni correction. *P*-values were capped to 1, if adjusted *P*-values were larger than 1 (i.e. ' $P=1$ '). In case of a significant main effect of condition, or any interaction between condition and another variable, each condition was compared to control, and LBP and CalFP were compared to each other as appropriate. Data and ANOVA residuals were checked visually for normal distribution using QQ plots and Shapiro Wilk tests for normality. Data were log-transformed if not normally distributed, consequently SDs were log transformed.

3. Results

3.1. Pain intensity

During LBP, the average pain intensities were 4.9 ± 1.7 and 4.5 ± 2.1 at 0.94 ms^{-1} and at 1.67 ms^{-1} , respectively. During CalFP,

Table 1
Results of repeated measures ANOVA of temporal gait parameters.

Variable	Condition		Speed		Condition × speed		Side		Condition × side	
	F-value	P-value	F-value	P-value	F-value	P-value	F-value	P-value	F-value	P-value
Stride time	11.07	< 0.001	426.06	< 0.001	4.03	0.01	–	–	–	–
Stance time	9.26	< 0.001	453.17	< 0.001	6.03	< 0.0001	0.16	0.69	7.70	< 0.0001
Swing time	11.22	< 0.001	236.57	< 0.001	1.86	0.13	0.17	0.69	7.78	< 0.0001
Condition × speed post-hoc						Condition × side post-hoc				
	<i>Speed 1 (0.94 ms^{−1})</i>					<i>Right leg</i>				
	Control vs.				LBP vs. Calfp	Control vs.				LBP vs. Calfp
Variable	LBP	wo LBP	Calfp	wo Calfp		Variable	LBP	wo LBP	Calfp	wo Calfp
Stride time	< 0.001	0.46	< 0.001	0.26	1	Stance time	< 0.0001	1	< 0.001	0.44
						Swing time	1	< 0.001	1	0.01
	<i>Speed 2 (1.67 ms^{−1})</i>					<i>Left leg</i>				
	Control vs.				LBP vs. Calfp	Control vs.				LBP vs. Calfp
Stride time	LBP	wo LBP	Calfp	wo Calfp		LBP	wo LBP	Calfp	wo Calfp	
	0.82	1	0.001	1	0.24	Stance time	< 0.0001	0.71	< 0.001	0.07
						Swing time	< 0.0001	0.01	< 0.001	0.06
						<i>Right leg vs. left leg</i>				
						Control	LBP	wo LBP	Calfp	wo Calfp
						Stance time	0.01	1	0.07	< 0.001
						Swing time	0.01	1	0.07	< 0.001

The *F*-statistics and corresponding *P*-values of temporal gait parameters and post-hoc tests with Bonferroni correction are reported for low back pain (LBP), washout (wo) LBP, calf pain (CalFP) and wo CalFP. Note that no post-hoc was performed on the significant condition × speed interaction of both stance- and swing time as these interactions are linked to the significant condition × speed interaction of stride time. The condition × speed × side of stance- and swing time were not significant ($F < 1.84$, $P > 0.13$) and is therefore not reported this table. Significant *P*-values ($P < 0.05$) are highlighted in bold.

the average pain intensities were 5.4 ± 1.5 and 4.8 ± 1.9 at 0.94 ms^{-1} and at 1.67 ms^{-1} , respectively. Pain intensity was not significantly different between locations ($F=2.00$ and $P=0.18$), but was lower at 1.67 ms^{-1} than at 0.94 ms^{-1} ($F=4.49$ and $P=0.05$). Pain was restricted to the area around the site of hypertonic saline injection.

3.2. Temporal gait parameters

3.2.1. LBP

Stride time was shorter during LBP than control at 0.94 ms^{-1} , but not at 1.67 ms^{-1} (see Table 1 for *F*-statistics and corresponding *P*-values (Fig. 1)). Independent of speed, stance time of both legs was shorter during LBP than control. Swing time of the left, but not right leg was shorter during LBP than control. Stance and swing time were not significantly different between left and right legs during LBP.

During washout LBP, stance time of both legs was not significantly different from control, but swing time duration of both legs was longer than control.

3.2.2. CalFP

Stride time was shorter during CalFP than control at both speeds (Fig. 1) and was not significantly different from control during washout CalFP. Left and right leg (painful leg) stance time and swing time of the left but not right leg was significantly shorter during CalFP than control. During CalFP, right leg stance time was shorter and swing time was longer than the left leg. Together, these findings can be interpreted as “limping” during CalFP.

During washout CalFP, stance time was not significantly different from control of either leg, but right leg swing time was longer. During control, right leg stance time was longer and right leg swing time was shorter than the left leg. The use of the right leg for recording of muscle activity may have contributed to this observation and may imply that we underestimated the effect of CalFP on this parameter.

3.3. Thorax movement

3.3.1. Maximum Lyapunov exponent (LDE)

3.3.1.1. *LBP*. During LBP, independent of movement axes, gait stability was lower (LDEs were higher) than control when participants walked at 0.94 ms^{-1} , and LDEs were similar to control values during washout LBP (see Table 2 for *F*-statistics and corresponding *P*-values (Fig. 2)). In contrast, at 1.67 ms^{-1} gait stability was higher (LDEs were lower) during both LBP and washout LBP than control.

3.3.1.2. *CalFP*. With CalFP, gait stability was lower (LDEs were higher) than control at 0.94 ms^{-1} (Fig. 2), but CalFP did not affect LDE significantly when participants walked at 1.67 ms^{-1} . At both speeds, LDEs were not significantly different from control during washout CalFP. Gait stability was lower (LDEs were higher) with CalFP than LBP at both walking speeds.

3.3.2. Magnitude and variability

3.3.2.1. *LBP*. LBP did not significantly affect thorax RMS along any of the axes (Table 2 and Fig. 3). However, RMS along the VT axis was higher during washout LBP than during control.

Along all axes at 0.94 ms^{-1} , SDs were greater during LBP than control (Table 2 and Fig. 3), and were not significantly different from control during washout LBP at this speed. In contrast, at 1.67 ms^{-1} SDs were lower than control during both LBP and washout LBP.

3.3.2.2. *CalFP*. With CalFP, independent of speed, thorax RMS along the ML axis was larger than control (Table 2 and Fig. 3), but was not significantly different from control during washout CalFP. RMS along AP and VT axes was not affected by CalFP. However, during washout CalFP, RMS along both of these axes was larger than control. RMS along the ML axis was larger with CalFP than LBP. RMS along the AP and VT axes were not significantly different between the pain conditions.

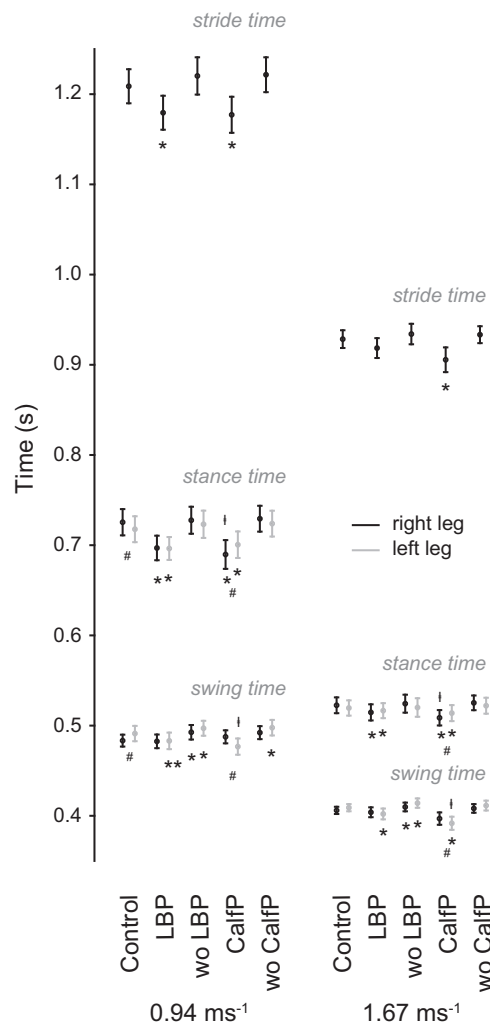


Fig. 1. Temporal gait parameters. Stride, stance and swing time are shown for control, low back pain (LBP), washout (wo) LBP, calf pain (CalFP) and wo CalFP. * – significant ($P < 0.05$) differences from control; # – significant differences between left and right leg; ♦ – significant differences between LBP and CalFP. Note that the asymmetrical change in stance time (decreased on painful leg) suggests limping with CalFP. Mean \pm SEM are shown.

Along all axes, SDs were also larger during CalFP at 0.94 ms^{-1} than control and were not significantly different from control during washout CalFP. At 1.67 ms^{-1} , SDs were not affected significantly by CalFP, but they were smaller during washout CalFP than control at this speed. Overall, SDs were larger during CalFP than LBP at both speeds.

4. Discussion

Partially consistent with our first hypothesis, the results of this study show that nociceptive irritation of a calf or back muscle reduces gait stability at low walking speed. Consistent with the second hypothesis, the results show that the effects of pain on gait stability are larger for calf pain than LBP. Somewhat unexpectedly, these effects were not found at high walking speed, and for LBP even reversed. These differences might be explained by different objectives of motor adaptation with different tasks and differences in biomechanics.

4.1. Why does pain affect gait stability?

Broadly, changes in movement during pain have been considered to reflect either adaptations that serve to protect the painful tissue, or arise from negative consequences secondary to the nociceptive stimulation. In both cases, the expression of adaptation is likely to be molded by pain intensity, past experiences, perceived threat, pain beliefs, context and task constraints (e.g., Hodges and Tucker (2011) and Moseley and Arntz (2007)). In the case of the former, it is assumed that adaptations will modify load on the painful structure (e.g. limit movement amplitude, or contraction intensity). In the case of the latter, mechanisms at multiple nervous system levels enable pain to interfere with motor function, including the effects of nociceptor activation on motor-neurons (Iggo, 1961; Paintal, 1960) and effects at the motor cortex (Martin et al., 2008; Tsao et al., 2008). Changes in gait stability observed in the present study can be interpreted with respect to these different processes. This interpretation is not straightforward and depends on the location of pain (injected muscle) and gait speed.

4.2. Effect of LBP on gait stability

LBP changed gait stability at both walking speeds, but the effect was opposite for each. The difference between speeds might be explained by different values placed on the pain in each context. For instance, at the faster speed, where forces and muscle activation are greater, and the potential consequence of perturbations is also greater, pain may lead the nervous system to optimized control of gait stability. At the slower speed where the potential consequence of perturbation is less, pain may be less important and induce less adaptation of control. This hypothesis is supported by the observation of greater variability at the slower speed, but less variability at the faster speed during LBP. Lower thorax variability during LBP at the faster speed might reflect a protective neuromuscular control strategy with the objective to enhance attenuation of perturbations between the pelvis and thorax, as reflected by the greater gait stability, potentially as a result of enhanced trunk stiffness. Ross et al. (2015) did observe a positive relation between trunk stiffness and stability (LDE) of spinal movements during a flexion-extension task, however on average trunk stability decreased with pain in this study. Increased gait stability with LBP at the faster speed could lead to more predictable trunk movements. This might be necessary to compensate for the potential of altered proprioception due to pain (Matre et al., 2002), and/or less effective corrective strategies (Mok et al., 2007). Although increased stiffness may be successful for control of small amplitude perturbations experienced in the predictable task of treadmill walking as tested here, it may limit the potential for control and recovery from larger perturbations (Mok and Hodges, 2013).

4.3. Effect of calf pain on gait stability

In line with our hypothesis, CalFP reduced gait stability at the slower speed. Further, at the slower speed CalFP affected gait stability more than LBP. Adaptations to pain depend on the muscle that is the source of nociceptive input (Hug et al., 2014). Because of the critical role they play in gait, any adaptation to calf muscle function is likely to have a greater effect on gait features, such as gait stability, than changes to back muscles. In addition to their primary role in propulsion, calf muscle activity is a key determinant of walking speed, vertical support (Anderson and Pandey, 2003; Ellis et al., 2014) and mediolateral balance (Kim and Collins,

Table 2
Results of repeated measures ANOVA of thorax movements.

Variable	Condition		Speed		Axis		Condition × speed		Condition × axis		
	F-value	P-value	F-value	P-value	F-value	P-value	F-value	P-value	F-value	P-value	
LDE	3.20	0.02	15.21	0.001	31.26	< 0.001	4.55	0.003	1.53	0.15	
RMS	5.18	0.001	163.14	< 0.001	67.42	< 0.001	3.53	0.01	6.57	< 0.001	
SDs	2.15	0.09	167.00	< 0.001	2.82	0.08	2.50	0.05	1.71	0.10	
Condition × speed post-hoc						Condition × axis post-hoc					
<i>Speed 1 (0.94 ms⁻¹)</i>						<i>AP movement axis</i>					
Control vs.						Control vs.					
Variable	LBP	wo LBP	CalFP	wo Calfp	LBP vs. CalFP	Variable	LBP	wo LBP	CalFP	wo Calfp	LBP vs. CalFP
LDE	0.04	0.74	< 0.001	0.36	0.01	RMS	1	0.29	1	0.003	0.32
SDs	< 0.001	1	< 0.001	0.24	< 0.001						
<i>Speed 2 (1.67 ms⁻¹)</i>						<i>ML movement axis</i>					
Control vs.						Control vs.					
Variable	LBP	wo LBP	CalFP	wo Calfp	LBP vs. CalFP	RMS	LBP	wo LBP	CalFP	wo Calfp	LBP vs. CalFP
LDE	0.001	0.001	1	0.09	< 0.001		1	1	< 0.001	1	0.04
SDs	0.02	< 0.001	1	0.02	0.002						
						<i>VT movement axis</i>					
						Control vs.					
						RMS	LBP	wo LBP	CalFP	wo Calfp	LBP vs. CalFP
							1	< 0.001	1	< 0.001	0.40

The F-statistics and corresponding P-values of Local Divergence Exponent (LDE), Root Mean Square (RMS) and magnitude of the stride-to-stride variability (SDs) from thorax movements (velocity) are reported for the anteroposterior (AP), mediolateral (ML) and vertical (VT) axes and post-hocs with Bonferroni correction for low back pain (LBP), washout (wo) LBP, calf pain (CalFP) and wo CalFP. Note that no post-hoc was performed on the significant RMS condition × speed interaction as the effect of the condition × axis interaction was greater. The condition × speed × axis was not significant in any of the thorax movement measures ($F < 1.76$, $P > 0.09$) and is therefore not reported this table. Significant P-values ($P < 0.05$) are highlighted in bold.

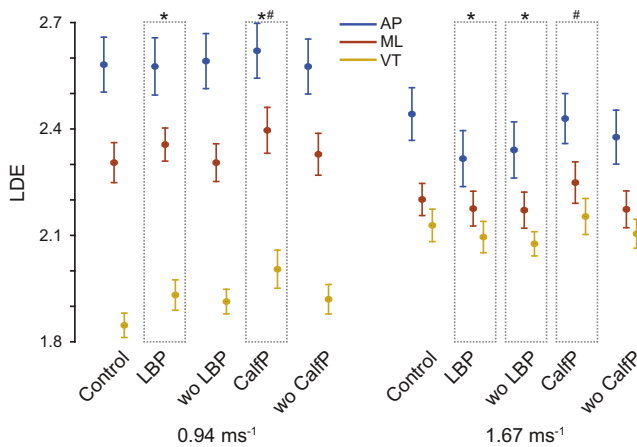


Fig. 2. Gait stability. The local divergence exponent (LDE) of thorax velocity along the anteroposterior (AP, blue), mediolateral (ML, red) and vertical (VT, yellow) axes are shown for control, low back pain (LBP), washout (wo) LBP, calf pain (CalFP) and wo CalFP. All differences were independent of movement axis which is highlighted by the dashed boxes. * – significant differences ($P < 0.05$) from control; # – significant differences between LBP and CalFP. Mean \pm SEM are shown. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

2015). Any adaptation, even subtle adaptation, could impact the overall gait pattern. This is demonstrated by the distinct “limping” during calf pain (e.g. asymmetrical change in stance time; decreased on painful leg), greater thorax RMS along ML axis and stride-to-stride thorax variability along all axes than both control and LBP at this speed.

At the slow speed, gait adaptations during CalFP are consistent with a protective solution to decrease tension in the calf muscle. Calf muscles contribute to swing initiation (Neptune et al., 2001), and reduced activity would explain the changes we observed. Although, these changes would disrupt gait, these could, to some extent be compensated by altered motion between the pelvis and thorax. For instance, data from an aligned study showed greater

hip flexor muscle activity and greater flexion-extension ROM between the pelvis and thorax in association with reduced calf muscle activity and limping (van den Hoorn et al., 2015). Although such changes may retain overall task objective, our data suggest that these adaptations reduced gait stability.

Contrary to our hypothesis, CalFP did not affect gait stability at the faster speed. Although thorax velocity along ML was greater during CalFP, and features consistent with limping were observed, thorax variability and stability were not affected. This implies that a different adaptation was adopted at the faster speed. Consistent with earlier arguments, this could be explained by the tighter constraint of walking at this speed, secondary to the greater potential for task failure from even minor disturbances. Other data support the tighter constraint of gait at faster speeds. For instance, inter-limb coordination improves with speed and has been related to improved ability to recover after perturbations (Krasovsky et al., 2014). Perturbations assessed in our study are small naturally occurring disturbances and are distinctly different from the large trip-inducing perturbation used by Krasovsky et al. (2014), therefore direct comparison is difficult. Although we imply enhanced active control of perturbations, simple mechanics could also explain the results. For instance, the greater momentum of the faster moving limbs could improve attenuation of perturbations, and the greater relative contribution of the force generated by the release of the stored energy in the muscle–tendon complex (passive recoil) at faster walking speeds (Hof et al., 1983; Lai et al., 2015; Lichtwark et al., 2007) could lead to a reduced sensitivity of gait kinematics to changes in calf muscle activation. Taken together, greater demand for tighter control of gait and the beneficial effect of changed mechanics at faster speeds could explain why CalFP did not have major impact on gait stability.

4.4. Implications

Reduced gait stability at lower speeds with experimental pain could have implications for older people at high risk of falls. Musculoskeletal pain in this population has been linked to falls (Asai et al., 2015; de Zwart et al., 2015; Kitayuguchi et al., 2015), and the

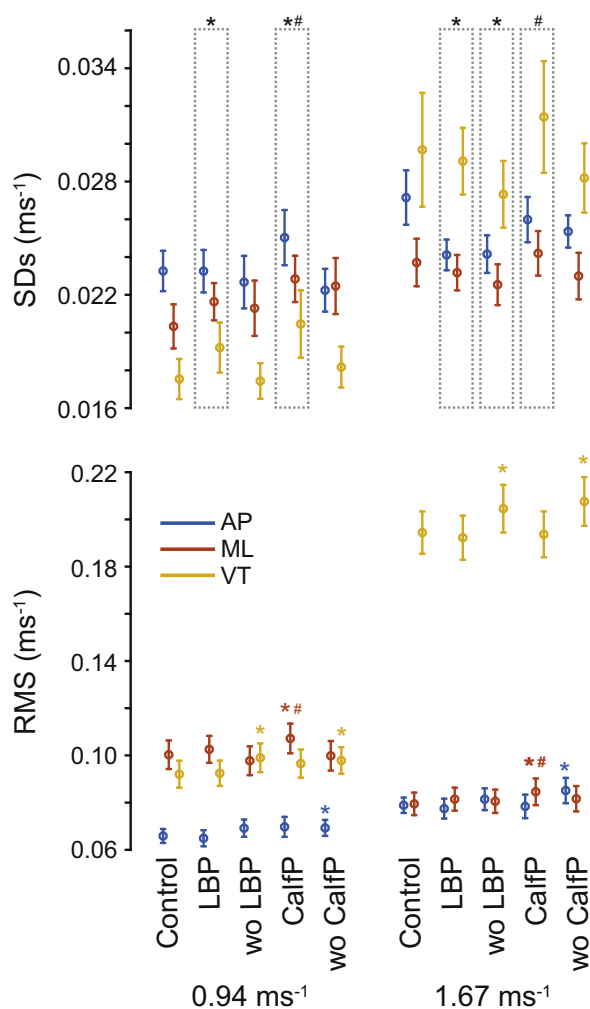


Fig. 3. Thorax variability and magnitude. The stride-to-stride variability (SDs) and root mean square (RMS) of thorax velocity along the anteroposterior (AP, blue), mediolateral (ML, red) and vertical (VT, yellow) axes are shown for control, low back pain (LBP), washout (wo) LBP, calf pain (CalFP) and wo CalFP. For SDs: the results were independent of movement axis, which is highlighted by the dashed boxes. * – significant ($P < 0.05$) differences from control; # – significant difference between LBP and CalFP. For RMS: the results were dependent on movement axis: * – significant differences from control; # – significant difference between LBP and CalFP (color of * and # show the difference for the respective axis). Mean \pm SEM are shown. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

results of the present study imply this might, at least in part, be explained by the negative effect that musculoskeletal pain has on gait stability. Although the average age of participants in the current study was young and pain was induced experimentally, it allowed examination of the effect of pain in isolation. Many factors could contribute to reduced gait stability in the elderly and musculoskeletal pain might be one of these factors. Future investigations are needed to investigate the potential relationship between muscle pain, gait stability and falls risk in older people.

Conflict of interest statement

The authors declare no conflicts of interest, financial or otherwise.

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